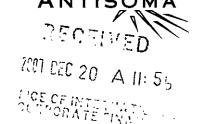
Press releases

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ASH presentation supports Antisoma's AS1411 phase II trial in AML

London, UK, and Atlanta, GA: 10 December 2007 – Positive preclinical data supporting Antisoma's ongoing phase II trial of AS1411 in AML (acute myeloid leukaemia) were presented this weekend at the American Society of Hematology (ASH) meeting in Atlanta, Georgia. Researchers from the Medical University of South Carolina showed that AS1411 killed AML cells with high potency and associated this with down-regulation of the apoptosis-blocking protein Bcl-2. Apoptosis is the programmed cell death pathway that normally removes malfunctioning cells.

The target of AS1411 is nucleolin. In AML cells, one function of nucleolin is to stabilise the messenger RNA for Bcl-2. This ensures high levels of Bcl-2 protein are maintained, placing a block on apoptosis. AS1411 binds nucleolin, leaving less available to stabilise the Bcl-2 message. Bcl-2 message levels therefore fall, leading in turn to a fall in the level of Bcl-2 protein. With the block on apoptosis removed, the cancer cells die. This helps to explain the powerful effect of AS1411 on AML cells.

Bcl-2 has been implicated in the resistance of some cancers to chemotherapy. It is therefore a desirable cancer target, but one that has proved difficult to exploit. AS1411 has potential advantages over approaches based on interfering directly with Bcl-2. First, it acts indirectly via a cell-surface target, avoiding any difficulty in accessing Bcl-2-related targets inside the cell. Second, its effect on Bcl-2 is part of a broader action that also includes interference with other functions of nucleolin in the cancer cell.

Professor Daniel Fernandes, leader of the group behind the ASH presentation, said: "Bcl-2 is of fundamental importance to the survival and proliferation of various cancers, and it is very interesting to see the high sensitivity of AML cells to AS1411 correlated with the drug's effect on this pathway. This accords with our previous findings with AS1411 in breast cancer cells."

Antisoma is conducting a randomised phase II trial of AS1411 in AML. This is evaluating the addition of AS1411 to standard therapy with cytarabine. A second phase II trial is planned in renal cancer, and plans for a third indication are being developed.

Glyn Edwards, Antisoma's CEO, said: "The ASH findings provide further support for the ongoing phase II trial of AS1411 in AML, which will report initial results in the first half of next year. The effect of AS1411 on Bcl-2 is of great interest, and is being taken into account as we select additional phase II indications for this very promising drug."

The poster presented at the ASH meeting is available on Antisoma's website at www.antisoma.com

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differ materially.

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**Notes for Editors:** 

Background on AS1411

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Aptamers are short pieces of DNA or RNA that can fold into stable, three-dimensional structures capable of interacting with particular target proteins. AS1411 is the first aptamer to be tested as a treatment for cancer. It binds to the protein nucleolin, which is found on the surface of cancer cells. It is then internalised and has been shown to kill cancer cells from a variety of cell lines. The drug has also shown anti-cancer effects in animal models and promising signs of anti-cancer activity in the clinic. AS1411 was originally developed by Dr Paula Bates, Dr John Trent and Prof. Donald Miller at the University of Alabama and then at the University of Louisville. Antisoma added AS1411 to its pipeline when it acquired the Louisville-based company Aptamera Inc. in February 2005.

## **Background on Antisoma**

Headquartered in London, UK, Antisoma is a biopharmaceutical company that develops novel products for the treatment of cancer. Antisoma fills its development pipeline by acquiring promising new product candidates from internationally recognised academic or cancer research institutions. Its core activity is the preclinical and clinical development of these drug candidates. Please visit www.antisoma.co.uk for further information about Antisoma.

